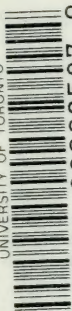


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Report on an outbreak of  
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REPORTS  
ON  
PUBLIC HEALTH AND  
MEDICAL SUBJECTS.

No. 20.

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REPORT ON AN OUTBREAK OF  
DYSENTERY

IN THE URBAN DISTRICT OF LYNTON, DEVON,

By

GEORGE C. HANCOCK, C.B.E., M.R.C.S., L.R.C.P.,  
*A Medical Officer of the Ministry of Health,*

and

P. BRUCE WHITE, B.Sc.,  
*Pathological Department, University of Bristol.*



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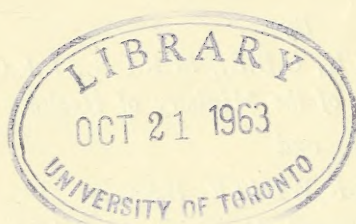


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## PREFATORY NOTE BY THE CHIEF MEDICAL OFFICER.

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To the Right Hon. NEVILLE CHAMBERLAIN, M.P.,  
Minister of Health.

SIR,

I BEG to present the following report on an outbreak of eighteen cases of Dysentery in the Urban District of Lynton, Devon, by Dr. Hancock, one of the Medical Officers of the Ministry, and Mr. Bruce White.

The nature of the illness was at first obscure, but on investigation it became evident that the symptoms presented by the sufferers were those of dysentery, and this was subsequently confirmed by full and complete bacteriological evidence. Infection was spread through the agency of milk, and the source of infection was traced to an active carrier of dysentery, a dairy foreman who had suffered an attack of the disease in 1918. This man, who had taken an active part in the milk production of the farm in question, was proved to be a carrier of mixed dysenteric infection by the isolation of *B. dysenteriae* Shiga and *B. dysenteriae* Flexner Z.

In January, 1919, the Ministry issued the Public Health (Pneumonia, Malaria and Dysentery) Regulations to meet contingencies of this kind, and it was practicable under these regulations to prohibit the dairy foreman from taking any further part in the dairy work or the distribution of the milk. As a result of the measures taken no further case of dysentery has occurred in the district.

This small outbreak is of interest not only because of the completeness with which it has been possible to co-ordinate the epidemiological and bacteriological findings, but also because of the special interest which attaches to these findings: dysentery is not commonly conveyed by milk, and the period during which the dairy foreman remained a carrier was exceptionally long.

I have the honour to be,  
Sir,  
Your obedient servant,

GEORGE NEWMAN.

Whitehall,  
*July, 1923.*



# REPORT ON AN OUTBREAK OF DYSENTERY IN THE URBAN DISTRICT OF LYNTON, IN THE COUNTY OF DEVON.

## PART I.

### EPIDEMIOLOGY.

BY G. C. HANCOCK.

The Ministry was informed on the 5th October 1922, by the Assistant Medical Officer of Health for the county of Devonshire that an outbreak of gastro-enteritis of obscure origin had occurred at Lynton, and asking for the assistance of a Medical Officer of the Ministry to investigate the occurrence.

As it seemed likely that the outbreak might be associated with some food material giving rise to illness, it was thought desirable to send a Medical Officer of the Foods Department of the Ministry, and I was accordingly instructed to visit Lynton, which I did on the 6th October, and commenced my inquiries the same evening.

Dr. Falkner, the Medical Officer of Health of the district, gave me the following particulars:—

He said that, during a period of three months commencing the 20th June 1922, 13 cases of illness marked by severe enteric symptoms had occurred in his practice and in the practice of Dr. Cummings, the other medical practitioner in the town. At no time had the outbreak shown explosive tendency, the cases cropping up at varying and irregular intervals, one in June, three in July, one in August and eight in September, and he was apprehensive of others occurring. The outbreak had been attended by an alarming fatality, no less than four deaths having occurred. The symptoms manifested by the sufferers were remarkably similar and characterised by diarrhoea accompanied by severe tenesmus and the passing of much mucus and blood in the stools. There had been comparatively little gastric disturbance and vomiting was absent except in some of the sufferers at the onset of illness. Fever had not been a marked symptom and in some of the severest cases the temperatures recorded were sub-normal throughout the greater period of illness. The enteric symptoms had shown in nearly all cases a high degree of severity, and in those which terminated fatally collapse, with its attendant phenomena, supervened early. The duration of illness had, in the majority, been 5 to 6 weeks except in those which terminated fatally. It had not been possible to determine the incubation period in the absence of known cause; the onset of illness was sudden and in only a few had there been any prodromal symptoms, these being marked by headache and a sense of malaise.



From the clinical symptoms presented by the sufferers it seemed unlikely that we were here dealing with an outbreak of food poisoning produced by an organism of the salmonella group. The absence of any marked gastric symptoms or of fever, the character of the stools—in many of the more severe cases little else than blood and mucus appears to have been passed *per anum*—and the long duration of the illness suggested an enteritis marked by dysenteric symptoms.

#### PREVALENCE OF DIARRHŒA AND DIARRHŒAL DISEASE IN RECENT YEARS.

Dr. Falkner, besides acting as Medical Officer of Health for the district, has been in practice at Lynton for nearly four years, during which time there appears to have been little prevalence of diarrhœa antecedent to the outbreak. Indeed, the district has enjoyed a remarkable immunity from diarrhœal disease and, with a single exception, no death has been recorded from this cause during the past four years. The single exception relates to a boy who visited Lynton with his parents in November 1921, and fell ill with symptoms similar in their clinical aspects to the group of cases which commenced on the 20th June 1922—seven months later. On the 14th November 1921, Dr. Falkner was sent for to see this boy. He had been ailing for a day or two beforehand, and when Dr. Falkner saw him he was suffering from profuse diarrhœa with blood and mucus in the stools. His illness ended fatally after four days' duration. The only other occupants of the lodging-house at the time of the boy's illness were the lodging-house keepers and the boy's parents. His father had only recently returned from Uganda, where he is said to have contracted malaria, but neither he nor his wife appear to have had dysentery. A number of holiday visitors had also stayed at the same lodging-house during the summer months, but no illness of any kind is said to have occurred among them.

Dr. Falkner was inclined to regard this boy as the source from which the subsequent series of cases originated; but if this were so it seemed difficult to understand why so long an interval as seven months should have elapsed between that fatal case and the first of the group of cases which began on the 20th June. On the other hand, it seemed clear that whatever was operating to produce this outbreak must have been intermittently operative for some time, for even in the group which had its beginning on the 20th June the cases were spread over a long period and cropped up at varying and irregular intervals.

#### STEPS TAKEN BY DR. FALKNER TO INVESTIGATE THE OUTBREAK PRIOR TO THE 5TH OCTOBER.

Dr. Falkner, with a view to determining the nature of the outbreak, procured specimens of the faecal discharges of one of the patients and sent it to Dr. R. V. Solly, who is in charge of the Pathological Department at the Royal Devon and Exeter Hospital. Dr. Solly reported on the 11th September, that he had

been unable to find evidence of any of the enteric or specific food poisoning organisms. Cultural tests were made for *B. dysenteriae*, *B. typhosus*, *B. paratyphosus* A and B; the food poisoning organisms—*B. enteritidis* Gaertner and *B. ærtrycke*, streptococci and the pathogenic anærobæ, but with negative results. The only organism found was *B. coli communis*. Examination for amœbæ and cysts also proved negative.

#### DESCRIPTION OF THE LYNTON DISTRICT AND DISTRIBUTION OF INVADÉD HOUSES.

Lynton occupies a site some 600 feet above sea-level and overlooks its sister village, Lynmouth, which lies at the bottom of a steep valley at the junction of the rivers Lyn. These villages, as well as the villages of Lynbridge and Barbrook, comprise the Urban District of Lynton. It has an area of 7,202 acres and a population of 1,770—1921 census. The number of occupied dwellings is 483. The rateable value is 10,868*l.* and 1*d.* rate produces 45*l.* 5*s.* 8*d.* The district is almost entirely residential, the inhabitants depending for the most part on the letting of "apartments" for a living. In the more remote parts of the district farming is carried on.

The outbreak has been confined to Lynton and, with a single exception, to a limited area of the town. No case has occurred at Lynmouth, Lynbridge or Barbrook. The single exception referred to relates to one of the later cases in the outbreak: a boy living on a farm two miles away, who was taken ill on the 19th October—a week after I had completed my preliminary investigations. The milk supply from this farm had from the first been suspect and under investigation.

#### THE SANITARY CIRCUMSTANCES OF THE DISTRICT CONSIDERED IN RELATION TO THE OUTBREAK.

Inquiry directed to the sanitary circumstances of the district failed to show any evidence that the water supply, the methods of excrement disposal, or the local sanitary circumstances of the area involved were responsible for the cases or for their continued prevalence.

*Water Supply.*—The Urban District of Lynton is provided with waterworks which supply water to a large majority of the inhabitants; only in the more remote parts of the district is water derived from springs. The Surveyor informed me that 379 dwellings were supplied with water from the former source and 104 from the latter, or 78 per cent. and 22 per cent. respectively. The waterworks were reconstructed in 1904, after local inquiry by Colonel A. G. Durnford, an engineering inspector of the Local Government Board. The water is derived from the river Lyn, the intake being situated about one mile above Lynton at a place called Barbrook Mills. Here it undergoes a preliminary filtration through sand for the purpose of arresting the larger



suspended matters. The water then flows by gravitation to two large filtering chambers known as Candy's Patent Oxidising Filters. Each of these contains four feet of filtering material composed of gravel polarite and sand. When filtering, the water enters at the top of the filter, passes through the bed of the filtering material and is discharged from the bottom of each filter through three outlets, each filter being divided by two vertical divisions into three compartments. They are cleaned by sending filtered water from one filter through the lower end of each compartment of the other filter which washes away the dirt collected during the filtering operation.

The maximum consumption of water is said to be about 100,000 gallons per 24 hours, which represents the total output of each filter, so that the two filters are quite capable of supplying the needs of the district at any time of the day. The supply is constant, but in order to guard against temporary shortage, a storage tank of 40,000 gallons capacity, which is filled directly from the filters, is available as a supplementary source of supply.

The gathering ground is moorland and rough pasture land, and the nearest dwelling above the intake is a single farmhouse which is situated about a mile from it. This farmhouse is undrained, human excrement being received into an earth closet and periodically disposed of by burial in farm land. Surface water from the farm premises at times, however, finds its way into the river owing to the blocking of a conduit which is intended to convey the surface water to meadow land on the north side of the farmhouse.

No case of diarrhoeal disease has occurred among the occupants of the farm, but the farm tenant who had been a sufferer from pulmonary tuberculosis for some years has recently succumbed to this disease.

Prior to 1903, the water appears to have been examined on several occasions and in that year Dr. J. O. Mitchell, the then Medical Officer of Health, reported: "With respect to the quality of the water I would refer you to the various analyses which have been made by Dr. Tidy, Dr. Wynter Blyth and Mr. Harland, each of whom characterised the water as of a very high order, and I may state that as far as I am aware no disease has ever arisen which can be directly attributed to pollution of the water." Analysis of the water recently made by Mr. Tickle, Public Analyst for Exeter City, shows that it is of high organic purity, and this despite the fact that the filters have not been recharged since they were installed in 1904. It should be mentioned in this connection that the Urban District Council have recently resolved to overhaul the installation, and to renew, if necessary, the filtering material.

As has already been mentioned, the outbreak has been confined to Lynton and to a limited area of that part of the district; Lynmouth, Lynbridge, and other parts of the district have escaped

altogether. There has thus been no generalised outbreak involving a number of the inhabitants such as commonly happens when infection is water borne; and the spread of the disease has been slow. Having regard to these facts and to the circumstances of the water supply, it seemed little likely that infection was conveyed by water.

*Sewerage.*—The whole of the district is sewered except Barbrook and farm lands situated on the outskirts. Lynton and Lynmouth are provided with 12-inch sewers, and Lynbridge with a 9-inch sewer. They are of stone-ware pipes with socket joints and converge to an outfall which discharges into the river Lyn at a point 480 feet from the sea at low-water mark. There is no separate system: all the sewers receive surface water as well as liquid refuse. The gradients are steep and there is abundant rainfall which serves to keep the sewers clean. As an additional precaution, however, flushing tanks are provided at three points, at or near to the heads of the sewers at Lynton, Lynmouth and Lynbridge. The sewers are unventilated except in the Lynton area, but the Urban District Council are considering the provision of extra ventilating shafts at the head of each sewer.

An objectionable feature of the sewerage system is the position of the outfall, the sewage being exposed for some four hours at each tide. At the point where the sewage discharges and for a considerable area surrounding it is a large collection of mussels and other shell-fish which have fastened upon the place as a suitable feeding ground. The part of the beach through which at low water the sewage with the waters of the Lyn flow seaward is frequented by the inhabitants as well as by visitors, especially in the summer months, and children are not seldom observed sailing their boats in the polluted waters of the river. In such circumstances an infected sewage might well prove a source of danger and inquiries were made to ascertain if any of the cases in the outbreak could be traced either to the consumption of mussels or to personal contact with the sewage effluent. In no case, however, had any of the sufferers eaten mussels gathered here, and only in one case did it seem at all likely that infection might have arisen by contact with the sewage effluent. The Urban District Council, in addition to other contemplated sanitary measures, have under consideration the question of extending the outfall further out to sea.

*House Drainage and Excrement Disposal.*—Nearly all the dwellings are provided with drains of sanitary type with socketed joints; also with proper gulleys, the area around which is usually paved or covered with impervious material. There are but few slop sinks, the water-closets being used mostly for this purpose. The latter are provided practically throughout the whole district and earth closets are only to be found in the neighbourhood of Barbrook. The water-closets are hand-flushed from flushing cisterns.



In the particular area of the district involved in the outbreak, the system of house drainage and excrement disposal is as described.

*Scavenging.*—House refuse is collected in pails which are placed by the inhabitants at the front or rear of their dwellings. The contents of these pails are collected three times a week by the Council's own staff. There are a few sanitary bins. All refuse is taken to a tip at Barbrook, at a distance of about 100 yards from the river Lyn, but considerably below the intake.

#### THE MILK SUPPLIES IN RELATION TO THE OUTBREAK.

There are 10 dairy farms registered under the Dairies, Cowsheds and Milkshops Orders for the sale of milk, most of which goes to Lynton. Lynmouth and other parts of the district obtain their supplies from farms in the Barnstaple Rural District.

In the course of my investigations the milk of a single farm situated some 2 miles from the village of Lynton came early under suspicion, for with two exceptions, every sufferer in the outbreak had consumed milk obtained from this farm, and even as regards the two excepted cases it cannot positively be stated that their illnesses were not associated with the implicated milk since the vendor of it, according to his own statement, had on occasions sold milk to other vendors whose supplies had run short on their rounds. It was ascertained that the farmer, an ex-Army officer, had served in the East during the war and had suffered an attack of dysentery in December, 1917, but had had no diarrhoeal attacks or symptoms of dysentery since then. He had also suffered repeated attacks of malaria and still has periodical manifestations of this disease. In March 1920, he started a livestock farm, but did not commence dairy farming until the middle of October, 1921. On the 14th November, some six weeks later, the first case to which reference has already been made occurred at the home of one of his customers—a lodging-house keeper at Lynton; and although no other similar occurrence of illness was reported until the 20th June, 1922, it was not without significance that this as well as nearly all subsequent occurrences were associated with the same milk supply. The farmer had taken an active part in the dairying and not only assisted in the handling of the milk, but himself distributed it to his customers. His milk round was confined to Lynton and bore a close correspondence to the area invaded; and of the 52 households which he supplied with milk, 9 or 17·3 per cent. were affected in the outbreak.

He had a pedigree herd of 17 cows and at the time of my visit these animals appeared to be in good health except one cow which had calved about three months beforehand and then developed inflammatory mastitis and indurated udder going on to abscess. Her milk had not been used for human consumption, but she had fed her calf, a strong, healthy young animal. In order

to make quite sure that the milk of this cow did not possess harmful properties, I obtained a sample of her milk as well as a sample of the mixed milk of the herd. Swabs were also taken from the affected quarter of the udder which was still discharging pus. These specimens, together with post mortem material from a child aged seven, were sent to Mr. Bruce White at the Department of Pathology, University of Bristol, in accordance with the arrangements made by the Ministry for bacteriological investigation in cases of food-poisoning (C.L.I., 10th August 1921). Other material sent to him at the same time were two specimens of fæces obtained from the farmer and his wife and one from a sufferer in the outbreak. He subsequently reported that he had been unable to find in any of the specimens sent to him any organisms of the dysentery or salmonella types.

As it seemed likely that the material had deteriorated in transit, Mr. Bruce White determined to visit Lynton and make further cultural experiments on the spot. He therefore visited Lynton on the 12th October and obtained fresh specimens of fæces from four convalescents and from one active case of the disease. Blood specimens were also obtained from these persons. The specimens of fæces were cultured there and then, and proved negative for pathogenic forms. The blood specimens failed to show significant agglutinations with any of the salmonella types or with such cultures of *B. dysenteriae* as were available at the laboratory. Later he received a specimen of blood taken from the farmer and also specimens of blood from five cows in his herd. These also proved negative.

So far then the most painstaking bacteriological investigations had failed to confirm the clinical manifestations of dysentery, and it was not until the 25th October that the true nature of the disease was revealed. On that date a specimen of mucoid fæces was received at Bristol University. It was derived from a fresh case of the disease occurring in a child aged six years, the son of the foreman employed at and residing on the farm under suspicion. From this specimen a *B. dysenteriae* Flexner was isolated without difficulty by the usual methods.

In view of these findings it was thought expedient that I should again visit Lynton in company with Mr. Bruce White in order to co-ordinate the necessary epidemiological and bacteriological work. We accordingly revisited Lynton on the 6th November and at once proceeded to the farm. Inquiry was then directed to the history of every person employed on the farm, and it was ascertained that besides the farmer, his foreman and two of the farm hands were ex-soldiers who had served in the East during the war, and two of them—the foreman and one of the farm hands—had been invalided with symptoms suggestive of dysentery. Specimens of blood were taken from these two men and a further specimen of blood from the farmer: two series of rectal swabs were also obtained in each case. The blood



specimens were examined against all the five main Flexner types and against the organism isolated from the foreman's child, and it was found that of these sera those of the farmer and his foreman gave indubitable positive reactions, while that of the farm hand gave a negative reaction. Examination of the rectal swabs proved negative in the cases of the farmer and the farm hand, but positive in the case of the foreman, two cultures of *B. dysenteriae* Flexner being obtained.

On the 8th November I was informed of another new sufferer in the outbreak. I visited him the same day with Mr. Bruce White, who took rectal swabs and mucus from his freshly voided faecal discharges. These were cultured on the spot and the culture plates subsequently developed a rich and almost pure growth of *B. dysenteriae* Shiga. It was unfortunate that this discovery was not made until after Mr. Bruce White's return to Bristol as serological examination at Lynton had centred on the Flexner types, and the blood of the farm-foreman and farm hand had not been examined for Shiga agglutinins. Meagre specimens from these men forwarded subsequently did not permit proper examination.

In the meanwhile arrangements had been made for the periodical examination of specimens of faeces obtained from the farmer and his foreman as well as from the two farm hands who had hitherto given no cultural or serological reactions. In this way it became possible to exclude the two latter from any concern in the outbreak. In the case of the foreman, however, exhaustive examination of three separate specimens of his stools definitely established a mixed infection by the isolation of *B. dysenteriae* Shiga and *B. dysenteriae* Flexner Z. In the case of the farmer a like examination was made of three separate stools from all of which organisms were isolated which on sub-culture were closely alike, and which also bore a near resemblance to *B. dysenteriae* Flexner. Mr. Bruce White does not, however, regard this organism as true *B. dysenteriae* and Dr. Scott, who has examined it at the Ministry's Laboratory, regards it as a variety of *B. dysenteriae* which is not true to type.

Besides the above bacteriological examinations blood specimens were obtained from four sufferers in the outbreak which serologically gave further confirmatory evidence as to the nature of the disease. The detailed results of Mr. Bruce White's bacteriological investigations are given in Part II. of this report.

#### SUMMARY OF CASES AND AGE INCIDENCE.

The total number of cases that have occurred since the commencement of the outbreak is 18, and the number of affected households 11. The age incidence has been chiefly on children, 13 occurring below the age of adolescence and five above that age. Multiple cases have occurred in five households and the sequence of attack in some of these suggests that case to case infection may have operated.

## STEPS TAKEN TO DEAL WITH THE OUTBREAK.

As soon as the nature of the outbreak and even before its cause was definitely established the farmer voluntarily withheld the sale of his milk and none was sold for human consumption after the 10th November. When the source of infection of the milk was finally determined a formal notice under Article XIII. of the Public Health (Pneumonia, Malaria and Dysentery, &c.) Regulations was served on the foreman. In the case of the farmer, having regard to the very reasonable and helpful attitude which he had taken throughout the inquiry, his assurance that he would take no further part in the production or distribution of the milk was accepted. The requirements of the Regulations in the one case and of the farmer's undertaking in the other have been complied with and no further case of the disease has been reported since the 16th November 1922.

## CONCLUSION.

This small outbreak is of interest from the completeness with which it has been possible to co-ordinate the epidemiological and bacteriological findings. These leave no doubt that infection was spread through the agency of milk and that the source of infection was an active carrier or carriers of disease. The history of the outbreak, its long duration, its spasmodic character and its abrupt termination on the withdrawal from the dairy farm of a proved carrier of dysentery support the view that periodic infection was conveyed by milk and in no other way. Other possible causes were kept in mind and investigated, but no evidence was forthcoming that they had any connection with the outbreak.

G. C. HANCOCK.

A. W. J. MACFADDEN,  
*Senior Medical Officer.*



## PART II.

## BACTERIOLOGY.

By P. BRUCE WHITE.

In presenting this report it will be convenient to maintain in part the sequence in which the various observations were made, and then to tabulate the findings as a whole.

The observations may be divided into—

- (1) Those made during the period 7th October, 1922, to 18th October, 1922.
- (2) Those made during the period 25th October, 1922, to 10th December, 1922.

*Period 7th October, 1922, to 18th October, 1922.*

On 7th October, 1922, Dr. Hancock warned me by wire of the despatch of post-mortem material from Lynton. This material stated to be from a child aged 7, was examined without delay on its arrival by passenger train. The organs submitted were: Stomach, spleen, portions of liver and large and small intestine. Of these, the large intestine alone exhibited definite pathological change. This was acutely congested, and there was much swelling of the mucous lining. Careful and protracted bacteriological examination, backed by animal experiments, failed to demonstrate the presence of any organism capable of causing the condition.

A specimen of fæces from another sufferer contained abundant mucus; but again cultural and animal experiments gave negative results.

On 10th October, 1922, I received the following specimens by post:—

- (1) Specimens of fæces from C.T. (farmer) and Mrs. C. T. of ——— Farm.
- (2) Specimen of fæces from an acute case, R.R.
- (3) Pus from udder of cow with mastitis.
- (4) Milk from (a) General supply, ——— Farm.  
(b) Cow with mastitis, ——— Farm.
- (5) Water from well on ——— Farm.

All specimens were submitted to a thorough examination for *Salmonella* types, and for *B. dysenteriae* without positive result.

The specimen of pus yielded a variety of septic cocci in culture.

A young rabbit injected, *per os*, with the fæces of R.R. developed mucoid stools, and died six days later. Death was probably due to coccidiosis: attempts to isolate a pathogenic bacterium from the stools and organs failed, as also did an attempt to propagate the condition among animals.

The specimens of faeces from C.T. and Mrs. C. T. were found negative for the cysts of *Entamoeba histolytica*.

Dissatisfied with these negative results, I decided to visit Lynton and make cultures and observations on the spot. It seemed possible that material was deteriorating *en route* to the laboratory, and it was necessary to exclude amoebic infection by the examination of fresh material.

During this visit (12th October 1922 to 13th October 1922), I obtained—

- (1) Fresh specimens of faeces from four convalescents, Mrs. M., J. M., R. R., and B., and from one doubtful case—P., of ten days standing.
- (2) Blood specimens from all the above—though that of B. was, unfortunately, broken in transit to Bristol.

The specimens of faeces were cultured on the spot and also examined for amoebæ. All examinations were negative for pathogenic forms.

The blood specimens, as well as a blood specimen received later from C. T., failed to show significant agglutinative action on any of the *Salmonella* types or upon such strains of *B. dysenteriae* as were available at this stage. The cultures of *B. dysenteriae* employed were old Shiga and Flexner strains maintained at the Pathology department of the Bristol University, and later work has shown the Flexner strain to be unsuitable for agglutination purposes.

During this period results were, therefore, entirely negative.

#### *Period 25th October to 10th December, 1922.*

This period opened with the receipt of a specimen of mucoid faeces from S. junior, child of the foreman (S. senior) of ——— Farm.

From this specimen a *B. dysenteriae* Flexner strain was isolated without difficulty by the usual methods. Using absorption technique and the standard sera and standard killed emulsions of *B. dysenteriae*, issued by the Standards Laboratory, Oxford, I found this strain approximated very closely to the *B. dysenteriae* Flexner "Z" type of Andrewes, *see* Table I. (a).

This important finding made a further investigation at Lynton advisable. I, therefore, spent the period 6th November, 1922, to 9th November, 1922, at that town, importing the necessary apparatus for cultural and agglutination work.

By arrangement my visit coincided with that of Dr. Hancock; this rendered a discussion of the epidemiological and bacteriological aspects possible.

Blood specimens were collected from the following :—

J. M. and R. R. (convalescent cases).

C. T. (farmer), S. senior (farm foreman) and V. T. (farm hand).



These specimens were examined against all the five main Flexner types of Andrewes (V.W.X.Y.Z.), and against the organism isolated from the child, S. junior.

The sera of R. R., J. M., and C. T. were also examined for Shiga agglutinins. The results of these tests are set out in Table II.

To summarise, the sera of J. M., R. R., C. T., and S. senior gave indubitably positive results with the "S. junior" strain and with the X, Y, and Z Flexner types; that of C. T. also acted distinctly on the V and W types. None of the Flexner strains employed reacted to the serum of "V.T."

The sera of R. R., J. M., and C. T. were found negative for Shiga agglutinins; in the case of V. T., as in that of S. senior, no examination was made on this occasion for Shiga agglutinins.

Two series of rectal swabs were obtained from C. T. and from S. senior and V. T. All the culture plates showed a few non-lactose fermenting colonies, which were subcultured for proper examination at Bristol. These later examinations proved negative save in the case of S. senior from whom cultures of *B. dysenteriae* Flexner type Z were obtained. These may be referred to as S. senior Flexner I. For serological identification of these strains, see Table Ib.

On 8th November, 1922, I went with Dr. Hancock to see a new case reported by Dr. Cummings. The man, W. N., was passing mucus and blood. Specimens of mucus and rectal swabs were taken and cultured immediately. The culture plates gave a rich and practically pure growth of *B. dysenteriae* Shiga, serologically identified as shown in Table I(c).

It was unfortunate that this discovery was not made until after my return to Bristol, as serological examination at Lynton had centred mainly on the Flexner types. At my request C. T. kindly sent me further specimens of blood from V. T. and S. senior. The specimens were very meagre and badly hæmolysed; they gave negative results at dilutions of 1/50 upwards for Shiga agglutinins.

Dr. Falkner kindly sent specimens of blood from two sufferers hitherto unexamined serologically, viz. :—"Mrs. M. F." and "B." Both these sera gave powerful Shiga reactions (see Table II.), but had no effect on any Flexner type employed.

Meanwhile, it had been arranged that C. T. should send specimens of fæces from himself, S. senior, V. T., and F. at intervals, wiring me the time they should arrive by passenger train. These specimens were most extensively examined at once on arrival. The results were as follows :—

*S., Senior.*

1st specimen.—Normal stool; negative for *B. dysenteriae*.

2nd specimen.—Soft stool with one large lump of mucus. From the mucus a Flexner strain similar to that already

isolated from this man was obtained, and also several colonies of *B. dysenteriae* Shiga. The latter are referred to as "S., senior, Shiga I."

3rd specimen.—Softish stool containing few strands of mucus. *B. dysenteriae* Shiga isolated (S., senior, Shiga II.).

The details of the serological identification of the strains "S. senior, Shiga I. and II." are given in Table I (c).

V. T. (3 specimens) and F. (2 specimens). Negative for *B. dysenteriae*.

#### C. T.

The three specimens were found negative for *B. dysenteriae*. From two of them a peculiar organism was isolated which, on account of its serological properties, came temporarily under suspicion as a member of the Flexner dysentery group.

Though Flexner X and Z sera agglutinated this organism very considerably, and though the homologous antiserum agglutinated X and Z Flexner strains to 50 or 100 per cent. of full titre, the cultural properties of "organism C. T." were such as to exclude it definitely from the dysentery group. In absorption tests no marked overlapping of absorptive qualities between this strain and the Flexner types was manifest.

### DISCUSSION AND SUMMARY.

The results obtained clearly demonstrate the fact that the outbreak at Lynton was due to *B. dysenteriae*. Further, the cases in which bacteriological and serological diagnosis have been established, fall into two series:—

#### 1. Due to *B. dysenteriae* Flexner:—

S., junior, R. R., and J. M. (To these are probably to be added J. R., brother of R. R., Mrs. M., mother of J. M., who suffered a transient attack and P., who worked for 10 days with diarrhoea. The two last-mentioned were found negative for Shiga agglutinins, but were inadequately examined for Flexner agglutinins.)

#### 2. Due to *B. dysenteriae* Shiga:

W. N., B., Mrs. M. F.

In no case has the simplicity of infection been taken for granted, and search for mixed infections and agglutinins has been made. No evidence of such has been found.

The number of proved cases under consideration is small, but it is interesting to find that in Series 1, S., junior, R. R., J. R. and J. M. are children and recovered: while the two adults, Mrs. M. and P., were mildly affected. On the other hand, the



three cases in Series 2, all recovering after severe attacks, are adults.

The presumption would seem to be that Shiga cases in children occurred before this investigation, and were fatal, while the Flexner infection did not cause severe symptoms in adults. Mrs. M. F. presumably contracted the disease from contact with a child who died; this would seem to indicate that this child also suffered from Shiga infection.

With regard to the inhabitants of ——— Farm: the farmer (C. T.) and his foreman (S., senior) came under especial suspicion as possible carriers; the farm hands (V. T. and F.) had but little contact with the milk and its receptacles. The blood of C. T., strongly agglutinated the type Flexner strain, but careful examinations of his stools have proved negative for *B. dysenteriae*.

The blood of S., senior, was also positive for Flexner agglutinins; an incomplete examination for Shiga agglutinins was negative. From the stools of this man, *B. dysenteriae* Flexner Z. was isolated on two occasions and *B. dysenteriae* Shiga on two occasions. We have here undoubtedly the source of the Shiga infections, and probably also that of the Flexner infections. With regard to the latter, however, it must be remembered that S., senior, had lived in intimate contact with his son, a Flexner case—which infection antedated the other can only be surmised.

In conclusion, I would say that the strains of *B. dysenteriae* isolated at Lynton have been re-examined by Dr. W. M. Scott, and also by Dr. P. Manson Bahr in conjunction with Colonel Marrian Perry who have expressed agreement on all points.

Dr. Manson Bahr has been good enough to point out to me the special interest of the findings:—

(1) that milk is an unusual medium for the dispersal of *B. dysenteriae*;

(2) that the period during which the farm foreman has presumably been a carrier of *B. dysenteriae* is of unusual length. This man was invalided out of the Army in 1918, after suffering in the East from symptoms suggestive of dysentery, and has not since suffered from diarrhoeal disease.

P. BRUCE WHITE.

TABLE I.

(NOTE.—The tests given below were performed with the standard killed emulsions and 100 standard units sera issued by the Standards Laboratory, Oxford. In this table, as in Table II., varying degrees of agglutination, ranging from complete agglutination to traces, still visible to the unaided eye, are indicated by the signs + + + +, + + +, + +, + and tr. The macroscopic method was employed with incubation for five hours at 52° C.)

(a) *Serological properties of the Flexner strain isolated from S., junior, acute case.*

Serum.	Organism agglutinated.	Serum Dilution.						
		1/20	1/40	1/100	1/200	1/400	1/800	Control
V	V	++++	++++	++++	++++	+++	tr/—	—
V	S. junior	++++	+	—	—	—	—	—
V absorbed by S. junior.	V	—	++++	++++	++++	+++/++	—	—
W	W	—	++++	++++	++++	+++	+	—
W	S. junior	++++	+++	+++	+	—	—	—
W absorbed by S. junior.	W	++++	++++	++++	++++	+++	tr.	—
X	X	++++	++++	++++	++++	++++	++	—
X	S. junior	++++	++++	++++	++++	+++	tr.	—
X absorbed by S. junior.	X	++++	++++	++++	+++	++	tr.	—
Y	Y	++++	++++	++++	++++	+++	++	—
Y	S. junior	++++	++	++	++	++	+/tr.	—
Y absorbed by S. junior.	Y	++++	++++	++++	+++	++	+	—
Z	Z	++++	++++	++++	++++	+++	+	—
Z	S. junior	++++	++++	++	++	++	+	—
Z absorbed by S. junior.	Z	++	+	—	—	—	—	—

(b) *Serological properties of Flexner strain isolated from S., senior, supposed carrier.*

Serum.	Organism agglutinated.	Serum Dilution.						
		1/20	1/40	1/100	1/200	1/400	1/800	Control
Z	Z	++	++	++	++	++/++	tr.	—
Z	S. senior	++++	++	++	++	+++	+	—
Z absorbed by S. senior.	Flexner I.	+	tr.	—	—	—	—	—
Flexner I.	Z							



TABLE I.—*continued.*(c) *Serological identification of Shiga strains from W.N., acute case, and S., senior, carrier.*

Serum.	Organism agglutinated.	Serum Dilution.					
		1/20	1/40	1/100	1/200	1/400	Control
Shiga - -	Shiga (standard) -	++++	++++	++++	++	—	—
Shiga - -	S. senior Shiga I. -	++++	++++	++++	+tr.	—	—
Shiga - -	S. senior Shiga II. -	++++	++++	++++	+	—	—
Shiga - -	W.N. -	++++	++++	++++	++	—	—
Shiga absorbed by S. senior Shiga I.	Shiga (standard) -	—	—	—	—	—	—
Shiga absorbed by S. senior Shiga II.	Shiga (standard) -	—	—	—	—	—	—
Shiga absorbed by W.N.	Shiga (standard) -	—	—	—	—	—	—

TABLE II.

AGGLUTINATION TESTS PERFORMED WITH SERA OF SUFFERERS  
AND OF POSSIBLE CARRIERS AT ——— FARM.(a) *Sera of Sufferers.*

Name of Donor.	Organism agglutinated.	Serum Dilution.					
		1/20	1/40	1/80	1/160	1/320	Control
A.R.	Flexner V. - -	—	—	—	—	—	—
	"    W. - -	Not done	—	—	—	—	—
	"    X. - -	Not done	+++	+++	+	—	—
	"    Y. - -	Not done	++++	+++	+	—	—
	"    Z. - -	++++	++++	+++	++	—	—
	S. junior - -	++++	++++	+++	++	—	—
J.M.	Shiga (standard) -	—	—	—	—	—	—
	Flexner V. - -	+	tr.	—	—	—	—
	"    X. - -	Not done	++++	++++	++++	+++	—
	"    Y. - -	Not done	++++	++++	++++	++++	—
	"    Z. - -	++++	++++	++++	++++	++++	—
	S. junior - -	++++	++++	++++	++++	++++	—
Mrs. M. F.	Shiga (standard) -	? tr.	—	—	—	—	—
	Flexner V. - -	—	—	—	—	—	—
	"    Y. - -	—	—	—	—	—	—
	"    Z. - -	—	—	—	—	—	—
	S. junior - -	—	—	—	—	—	—
	Shiga (standard) -	++++	+++++/	+++	++	tr.	—
L. - -	"    "	"    "	"    "	"    "	"    "	"    "	"    "
	Senior Shiga I. -	++++	++++	++	+	—	—
	Flexner V. - -	—	—	—	—	—	—
	"    Y. - -	—	—	—	—	—	—
	S. junior - -	—	—	—	—	—	—
	Shiga (standard) -	++++	++++	++++	++++	++++	—
S. senior Shiga I.	Shiga (standard) -	++++	++++	++++	++++	++	—
	S. senior Shiga I. -	++++	++++	++++	++++	++	—

TABLE II.—*continued.*  
(b) *Sera of possible Carriers.*

Name of Donor.	Organism agglutinated.	Serum Dilution.					
		1/20	1/40	1/80	1/160	1/320	Contr
C.T.	Flexner V.	+++/ ++++	+++	+	—	—	—
	„ W.	+++	++	+	—	—	—
	„ X.	++++	++++	++++	++++	++	—
	„ Y.	++++	++++	++++	++++	++	—
	„ Z.	++++	++++	++++	++++	+	—
	S. junior	++++	++++	++++	++++	++	—
	Shiga (standard)	tr./—	—	—	—	—	—
	S. senior	+	—	—	—	—	—
	Flexner V.	+	—	—	—	—	—
	„ X.	Not done	+++	++	+	—	—
V.T.	„ Y.	Not done	+++	++	tr.	—	—
	„ Z.	++++	++++	+++	+	—	—
	S. junior	++++	++++	+++	tr.	—	—
			1/50	1/100	1/200		
	Shiga (standard)		—	—	—		—
	Flexner V.	—	—	—	—		—
	„ W.	—	—	—	—		—
	„ X.	—	—	—	—		—
	„ Y.	—	—	—	—		—
V.T.	„ Z.	—	—	—	—		—
	S. junior	—	—	—	—		—
			1/50	1/100	1/200		
	Shiga (standard)		—	—	—		—



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